tion in general practice. None of the studies reviewed by Wyld are of the rigorous, highly numerate quality needed to convince government to invest even more money in general practice. If the College is as interested in counselling as it appears to be, would not the most appropriate action be to initiate a critical, academically respectable study of counselling in general practice? This would be one step towards determining the most appropriate method of caring for the reported one third of patients who consult with symptoms determined by psychosocial factors.

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References

Sir,

Am I alone in finding the concept of counselling as outlined in your leader unsatisfactory? (June Journal, p. 323.) It is unsatisfactory because I am supposed to ‘assist the person to live the life he has consciously chosen’ and yet I find these life-styles are so often untenable. Am I really expected to encourage the drug addict, the nymphomaniac, the homosexual, the alcoholic and every other social misfit to continue his lifestyle ‘without being more dependent than he wants to be upon the decisions of others’? Why should the choice be his? Don’t the rest of us who live fairly humid orthodox lives have the right to reject these characters?

As far as I am concerned, the liberalization of society has gone too far, and I am not prepared to accept this responsibility of counselling in the terms of this definition.

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Children with Yellowed Palms and Soles

Sir,

Further to the abstract (June Journal, p. 326), I wonder if carotenaemia has been considered as a cause for this.

This is common in West African children who consume the oil palm fruit which is rich in carotene. Palm oil can be obtained from supermarkets in the UK, where it is sold for cooking purposes. Consumption of palm oil can rapidly raise the serum carotene levels to give the characteristic changes in the colour of the palms of the hands and the soles of the feet.

I would suggest that the serum carotene levels in these children be measured.

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Paranoid Psychosis Associated with Zimeldine Antidepressant Therapy

Sir,

A 57-year-old depressed Caucasian woman was admitted to hospital after a moderate self-overdosage with diazepam. Previously she had responded to tricyclic antidepressant therapy for a depressive illness that followed bereavement. This treatment had been associated with side effects such as lassitude and an impaired ability to concentrate. On this occasion she was treated with zimeldine hydrochloride 200 mg each morning; she responded well with improved mood, sleep pattern, appetite and sociability.

On the 13th day she complained of nausea and slept only intermittently; on the 14th a mild transitory rash appeared on both arms and legs. On the 15th day her behaviour changed. For the first time she voiced delusions that she had venereal disease; she washed her mouth out repeatedly and accused a West Indian male staff nurse of proposing to her. She made unpleasant racial comments to him and developed auditory hallucinations; she was weepy and slept poorly.

On the 16th day she locked herself in the lavatory and later went to the male ward several times and physically attacked male staff and patients. She threw a metal wastepaper bin through the dormitory door window.

Treatment with zimeldine was stopped and she was given chlorpromazine intramuscularly. She remained restless and unpredictable throughout the 17th and 18th days and totally lacked insight into her behaviour.

Her mood and sleep pattern returned to normal on the 19th day—that is three days after the zimeldine had been discontinued. She had only the vaguest recollections of what had occurred. She has since remained in excellent spirits and was asymptomatic at follow-up three months later.

This patient had had a mild cerebral-vascular episode 11 years earlier but had recovered fully within 12 months. No signs nor symptoms of this were apparent on this admission. Investigations, including a chest X-ray, electrocardiogram, blood count, Wassermann reaction and chemical pathology were all normal. There was no past history of any psychotic illness.

There are many causes of psychiatric drug reactions. Tricyclic and monoamine oxidase inhibitor drugs are thought to activate latent schizophrenia rather than to be directly responsible for paranoid psychoses. Existing psychotic manifestations including mania and paranoid delusions may be exacerbated during tricyclic and tetracyclic antidepressant therapy and visual hallucinations have been reported in association with imipramine and amitriptyline. A genetic predisposition has to be considered as we elicited subsequently that the patient’s sister (a year older) had committed suicide in 1982 after recurrent psychiatric illness since 1964—in 1973 she had been described as having recurrent depression with psychotic features.

This case reports a likely association between a useful antidepressant drug (acting by inhibition of serotonin uptake) and a paranoid psychotic adverse drug reaction. Cautious consideration in using this preparation is suggested in depressed patients whose symptoms include a schizophrenic element, or whose close relative has displayed psychotic features and, perhaps, where there has been a definite cerebro-vascular episode in the past.

This reaction has been notified to the Committee on Safety of Medicines and the manufacturers have also indicated their concern and desire that any similar case be brought to their notice.

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References